October 11, 2021

Reply to Bird, Carlson, and Townsend

My perception is that the interview process here is unfair. Bird et al. have too much power. They can allege whatever they want without accountability (despite potential defamation), and I am stuck trying to rebut their claims without the luxury of cross examination. This also lets them generate new accusations that I must now deal with, this late into the investigation. Specifically, I counted seven new allegations from this interview that were not previously "on the table". Bird et al. also made several allegations that are wholly irrelevant as to whether I have committed academic misconduct.

Below is my rebuttal of issues raised in their interview.

1. P.3, 37, "…racist applications of genetic data". I object to this statement; my research is not racist, because I employ the same techniques used by medical researchers to study race/ethnic group differences in disease susceptibility, or in neurological traits like sleep depth. Is research on group differences in susceptibility to cancer, or to sleep related neurology, racist?

2. P.3, 47, the Pioneer Fund. I did not receive money from the Pioneer fund. However, John and I applied for funding from Richard Lynn to pay for John's CSU tuition. For the record, I did apply for a Pioneer Fund grant back in 2008, but it was not funded. Even if I were funded (in 2008 or now), how would this be evidence of academic misconduct?

3. P.3, 52. The Ulster Institute. I am not affiliated with the Ulster Institute, but if I were, how would this be evidence of academic misconduct?

4. P.4, 56. Authorship with Davide Piffer. I have no publications with Davide, despite the absurd claim that he is a "frequent co-author" with me. Even if I did co-author with Davide, how is this evidence of academic misconduct?

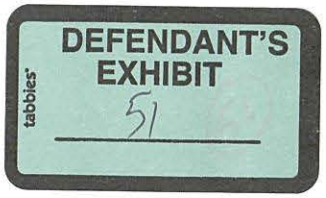
5. P.4, 64. ResearchGate Lab. How would having or not having a "lab" with John and Emil be evidence of academic misconduct? ResearchGate defines a "lab" as simply "a group of scientists, led by a senior researcher, who conduct experiments and research together on a specific topic."

6. P.4, 75. Fraudulent Listing of Lab Associates. The speculation here is inane and potentially defamatory.

7. P.5, 78. The Human Phenome Diversity Foundation. How is fundraising to support IQ research evidence of academic misconduct?

8. P.5, 88. Mankind Quarterly Publications. I am not an author on the "More research is needed" paper, and Google Scholar now reflects this.

9. P.6, 101. Uploading to the External Website. How is it possible that SNPs predicting just eye, hair, and skin color can be used to genetically identify this study's research participants?



10. P.7, 124. Explicit mention of Race/IQ in our Applications. Bird states: "I don't believe testing the racial IQ gap which separate points in the paper was explicitly kind of stated as the goal [of Pesta's applications] …". However, our Application (Transracial Validity of PGS) explicitly states: "The study will be a correlational analysis of the statistical association between various PGSs and cognitive ability. We will conduct analyses separately in White and African American samples. For these analyses, we will need cognitive data (all available) to create general and broad ability indexes, and demographic data (age, sex, etc.). We will also need SNP genotypes to create PGS scores and to control for genetic relatedness following the protocol of Lee et al. (2018)".

11. P.8, 127. Our Requests for More Datasets. How is also requesting the ABCD and Add Health datasets evidence of academic misconduct?

12. P.9, 147. Mention of Race and IQ in our Applications. We did, please see Point 10 above.

13. P.10, 149. Do our Newer Applications Mention Race/IQ? Bird says: "And the February 20 one, "Scarr-Rowe Effect on Genetic Expressivity" is about socio economic status. So, none of them, just from looking at them and from my recollection, mention testing a genetic racial cause of IQ differences." This is absurd. Our newer applications clearly state that our focus is on cognitive ability and race/ethnicity. We were rather careful with these newer applications, given that we had already received the 2019 complaint from the NIH. Again, despite the Bird et al. complaint in 2019, the NIH still approved our newer applications (leading us to believe that we were in the clear with them).

14. P.10, 154. Ethics of our Research. This is speculation by Bird. Why would the NIH approve our newer applications if studying "…genetic ancestry, education related PGS, and cognitive ability" (i.e., our ABCD Applications) is unethical?

15. P.10, 166. IRB Approval. I wish I were asked about IRB approval beforehand, rather than have Bird et al. speculate at length as to whether I have it. I do have it, for those applications where it was needed, and I attach the approval/exemption letter here. This further illustrates my frustration with the interviews—again, my perception is that these external people can say whatever they want (no cross examination), and I am stuck trying to defend all this, when they have no authority to make most of these kinds of determinations (e.g., whether I had IRB approval).

16. P.13, 208. Connor paper. I am not an author on this paper. Google Scholar now reflects this.

17. P.14, 222. External Sever Upload. We did upload files to this server. So did the paper referenced below, which was published after our (Lasker et al., 2019) paper. The paper below is high-profile and published in the collection of journals from *Nature*. For this paper, the NIH approved the authors use of the same external server that we used, for the same purpose that we used it for. Why?

https://www.nature.com/articles/s42003-020-01461-8/figures/3

Genetic ancestry, skin pigmentation, and the risk of cutaneous squamous cell carcinoma in Hispanic/Latino and non-Hispanic white populations

- Eric Jorgenson, Hélène Choquet, Jie Yin, Thomas J. Hoffmann, Yambazi Banda, Mark N. Kvale, Neil Risch, Catherine Schaefer & Maryam M. Asgari

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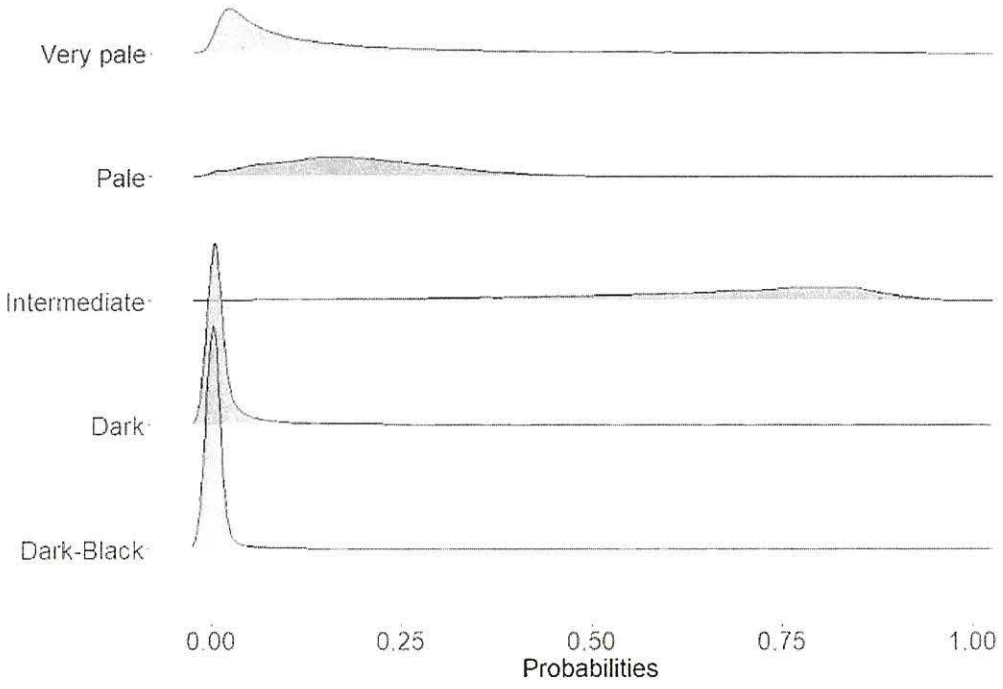
Data availability

Genotype data of GERA participants are available from the database of Genotypes and Phenotypes (dbGaP) under accession phs000674.v2.p2. This includes individuals who consented to having their data shared with dbGaP. The complete GERA data are available upon application to the KP Research Bank (https://researchbank.kaiserpermanente.org/).

HIrisPlex-S program and predicted skin pigments

To determine whether the genetic ancestry associations with cSCC were due to differences in skin pigmentation, we predicted skin pigment traits (i.e., "dark skin," "dark to black skin," "intermediate skin," "very pale skin," and "pale skin") using HIrisPlex-S24,25,26, a program that utilizes known pigment genetic variants to generate probabilities of different pigment traits. The HIrisPlex-S predictive model includes 36 SNPs for skin color (Supplementary Table 3). We did consider using a five-category level (very pale, pale, intermediate, dark, and dark-black) of the HIrisPlex-S skin pigmentation predictions and examined the distribution of all the categories in GERA non-Hispanic whites (Fig. 3). Because the distribution of the categories "very pale" and "dark to black" were not well differentiated from "pale" and "dark," respectively, we decided to combine "dark" and "dark to black skin" into a single category, and "very pale" and "pale" into a single category. Thus, by focusing on a three-category level (very pale + pale, intermediate, dark + dark-black), we improved the predictive values for skin pigmentation compared to the original five-category level. To determine whether predicted skin pigmentation traits explain the observed associations of genetic ancestry with cSCC risk, we included the probabilities for "dark + dark to black," and intermediate skin colors in the logistic regression models. Because the skin pigment probabilities sum to 1, we had to exclude at least one skin pigment probability. We chose to exclude "very pale + pale skin" from our models. As a result, the direction of the effect estimates of the skin pigment probabilities represents darker pigment.

Fig. 3: Distribution of the five-category level of the HIrisPlex-S skin pigmentation predictions in GERA non-Hispanic whites.



Skin pigment traits (i.e., "dark skin," "dark to black skin," "intermediate skin," "very pale skin," and "pale skin") were predicted using HIrisPlex-S program based on 36 SNPs known to influence skin color. We did consider using a five-category level (very pale, pale, intermediate, dark, and dark-black) of the HIrisPlex-S skin pigmentation predictions and examined the distribution of all the categories in GERA non-Hispanic whites.

Please note the additional inference here that if the NIH approved this paper, then obviously the external, HIrisPlex-S, website is not sufficient to genetically identify research participants.

18. P.15, 240. Complaints about Emil Kirkegaard. How is Emil's past research record (e.g., The OK Cupid study) relevant as to whether I committed academic misconduct here?

Reply to Kent Taylor's Interview

1. P.2, 44. The Journal, *Psych*. How are articles from other scholars, albeit edited by me, evidence of my academic misconduct? Moreover, regarding my "view" on whether genetics play a role in IQ / Race gaps, I am currently agnostic.

2. P.7, 127. "Outside the data use". I have no opportunity here to defend against this statement, as Taylor doesn't say why or how I am "outside the data use."

3. P.7, 32. Stigmatization. My understanding is that academic freedom trumps whether people find speech offensive (see the correspondence from FIRE in the binder), but I do not know if "stigmatization" is in a separate category, or merely an exemplar of how something might be offensive. I defer to Jay.

4. P.10, 177. "[my work] is clearly outside what you should be doing". Another reason why I think the process here is unfair is that I cannot call witnesses to rebut statements like these. I could call bona fide experts to testify that our research agenda was scientifically appropriate.

5. P.11, 192. "Not statistically high quality at all." Why? How? The statistical analyses (e.g., MGCFA) employed in Lasker et al. were a strength of the paper. If given the chance, I could readily defend the statistical expertise of all Lasker et al. co-authors, together with all analyses we conducted.

6. P.11, 192. "There's a lot of things on socio-economic status that are missing". We (Lasker et al.) controlled for SES in our analyses, which we suspect is obvious for people who have read our paper beyond the abstract. Moreover, we clearly note the limitations of our research design in Lasker et al., which are also commonly referenced in the medical epidemiology literature:

While the statistical mediation by PGS scores suggests that genetic factors may be involved, as discussed in detail by Kirkegaard et al. [24], **we cannot rule out many types of confounding environmental variables with this research design. Global admixture analysis results are suggestive and should only be considered a first step for investigating the effects of admixture on a trait**. We suggest to attempt replication of the current results using a nationally representative sample and then, if these findings are confirmed, proceed to admixture mapping (local admixture analysis). This is the standard approach taken in medical epidemiology.

Sincerely,

Bryan